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Address:
Senol Bozdag
Kizilirmak Mah. 48
Cadde 449
Sok No: 37/19 06300
Cankaya/Ankara
Turkey
Tel.: +90 505 691 76 65
Fax: +90 312 236 21 01
E–mail: senolbozdag@hotmail.com

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Case Report
TMJ Disorders

A case of destructive calcium pyrophosphate dihydrate crystal deposition disease of the temporomandibular joint: a diagnostic challenge

J. Meng[1], C. Guo[1], H. Luo[2], S. Chen[1], X. Ma[3]
[1]Department of Oral & Maxillofacial Surgery, Peking University School and Hospital of Stomatology, Beijing, China; [2]Department of Oral Pathology, Peking University School and Hospital of Stomatology, Beijing, China; [3]Center for TMD and Orofacial Pain, Peking University School and Hospital of Stomatology, Beijing, China

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Abstract. The authors present the case of a 64-year-old woman with a destructive calcium pyrophosphate dihydrate (CPPD) crystal deposition disease of the temporomandibular joint. Progressive pain, swelling and a malocclusion were her chief complaints. A few granular calcified masses surrounding the left condylar head and extending to the infratemporal fossa and middle cranial base were presented in CT images. It occurred alone without other joints being affected. A provisional diagnosis of occupying lesion with invasion was made preoperatively, but histologically, the mass contained numerous deposits of rod-shaped or rhomboid crystals, which were positively birefringent under a polarising microscope, suggesting a CPPD deposition disease. The histopathological diagnosis was further supported by scanning electron microscopy with energy dispersive X-ray spectroscopy. The diagnosis, differential diagnosis and treatment of this disease are discussed.

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Calcium pyrophosphate dihydrate (CPPD) crystal deposition disease is characterized by the accumulation of pyrophosphate dihydrate crystals in articular and periarticular tissues. Large joints such as the knee and wrist are the most common sites affected. CPPD deposition disease of the temporomandibular joint (TMJ) is rare[29]. The authors present the case of a 64-year-old woman with a destructive and invasive facial mass extending to the infratemporal fossa. A provisional diagnosis of an occupying lesion with invasion was made preoperatively.

Case report

A 64-year-old woman was admitted to hospital for the diagnosis and treatment of a swelling and pain in the left preauricular region. The patient presented a 5-year history of chronic pain and swelling. The

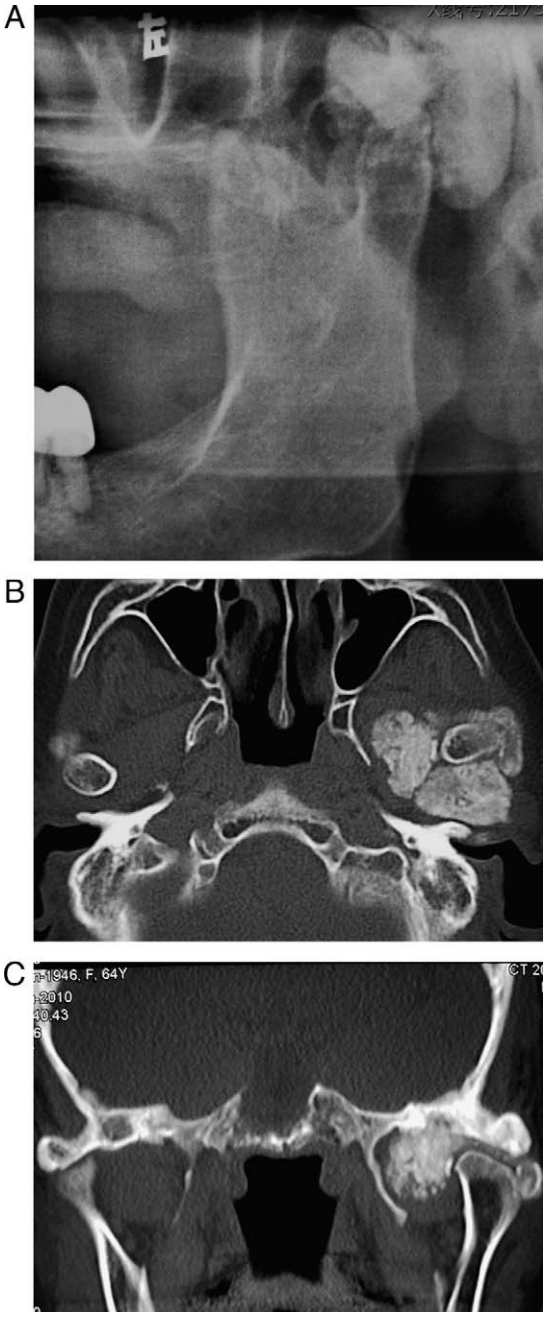

Fig. 1. (A) Panoramic image showing three large masses with gravel-like appearance and calcified foci in the left joint region. The glenoid fossa was destructive. (B) Axial CT scan showing a few granular calcified masses surrounding the left condylar head, and extending into the infratemporal fossa. (C) Coronal CT scan revealing calcified mass in the joint space. Destruction and sclerosis of the middle cranial base were present and the lesion seemed to extend into the middle cranial fossa.

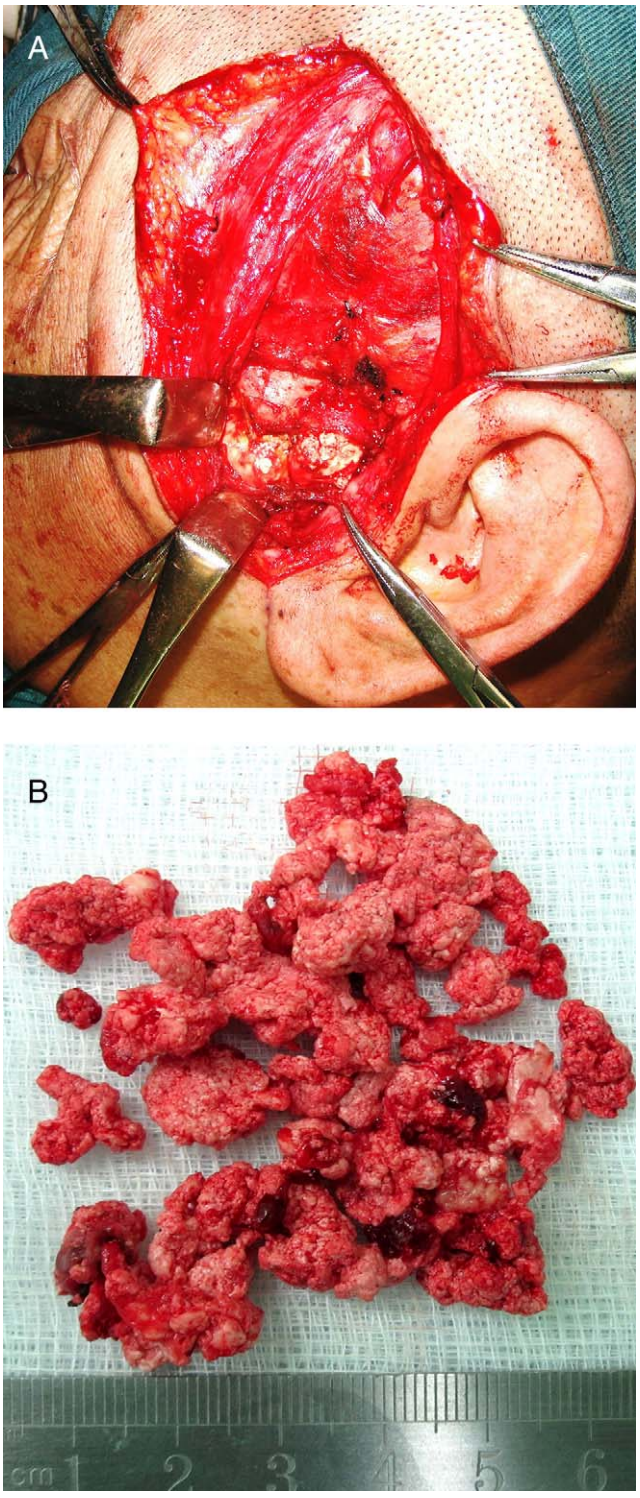

Fig. 2. (A) Excision of the lesion at surgery, a few white and gritty, dough-like masses were removed. (B) Photomicrograph of the specimen.

symptoms were aggravated and followed a malocclusion with a deviation of mandible and a limitation of mouth opening for 1 year. She had a history of hypertension, hypercholesterolaemia and a gallbladder stone. There was no history of trauma to the orofacial region and no history of metabolic disturbances, such as hyperparathyroidism, chronic renal failure or diabetes. There was no history of disease involving any other joints. The preoperative blood investigations showed slightly higher phosphate, 1.49 mmol/l (normal 0.81–1.46) and cholesterol 5.89 mmol/l (normal 3.10–5.70), but other electrolytes, including urate and calcium, were normal.

Clinical examination showed an obvious preauricular swelling with tenderness on the left side. Stenosis of the external ear canal was found because of the swelling, but there was no hearing loss. Interincisal mouth opening was limited to 25 mm. A malocclusion was present with a slight deviation of mandible to the right side. She had a maxillary complete denture and removable partial denture in the mandible with only eight teeth left.

A panoramic image showed three big masses with gravel-like appearance and calcified foci in the left joint region. The glenoid fossa was destructive and the condyle was displaced anteroinferiorly due to the extrusion of the masses (Fig. 1A). On an axial CT, there were a few granular calcified masses surrounding the left condylar head and extending into the infratemporal fossa (Fig. 1B). Coronal CT scans revealed calcified mass in the joint space. Destruction and sclerosis of the middle cranial base were presented and the lesion seemed to extend into the middle cranial fossa (Fig. 1C). Although sclerosis was shown in the condyle, the right joint had no evidence of a similar manifestation to the left side on the CT scan. A provisional diagnosis of occupying lesion with invasion such as chondroma/osteochondroma or chondrosarcoma/osteochondrosarcoma was made preoperatively.

Surgical exploration of the left TMJ was planned. During surgery, a few white and gritty, dough-like masses were removed from the lateral aspect of the upper joint space (Fig. 2). Microscopically, the frozen section revealed much crystalline material within the biopsy specimen. A large amount of similar white, gritty material was curetted from the anteromedial and posterior aspects of the upper joint space. Although the glenoid fossa was destructive, it was not perforated with the skull base. The meniscus appeared normal in colour and texture. No additional masses were found in the inferior joint space. There were no destructive changes in the condyle.

Histologically, under light microscopy, an amorphous substance or chondromyxoid tissue containing abundant crystal deposits was observed. The crystals were positively birefringent under a polarising microscope and rod- or rhomboid-shaped, which strongly suggested a diagnosis of CPPD deposition disease of the TMJ (Fig. 3).

Scanning electron microscopy (SEM) revealed rod- and rhomboid-like crystals mainly ranging from 1 to 5 μm in size. Energy dispersive X-ray spectroscopy (EDS) of these crystals showed peaks corresponding to calcium and phosphorus (Fig. 4). Qualitative analysis indicated that the calcium and phosphorus ratio was close to 1, which further supported the histological diagnosis of CPPD deposition disease.

The postoperative course was uneventful. The patient's clinical symptoms improved soon after surgery. Conventional radiographs of the knee and wrist were examined postoperatively and showed no similar image findings in these joints. The patient remained free of symptoms at the 8-month follow-up and had no evidence of recurrence.

Discussion

CPPD deposition disease is usually a benign condition characterized by crystal deposition of CPPD in synovial membranes, joint cartilages and surrounding soft tissues. CPPD deposition disease of the TMJ is rare; the disease is most commonly found in the knee joint. Other joints that may be affected are the wrists, elbows, shoulders and ankles. PRITZKER et al. first described it in the TMJ in 1976. It is also termed tophaceous pseudogout or chondrocalcinosis[1,29].

To the authors' knowledge, 42 cases of CPPD deposition disease affecting the

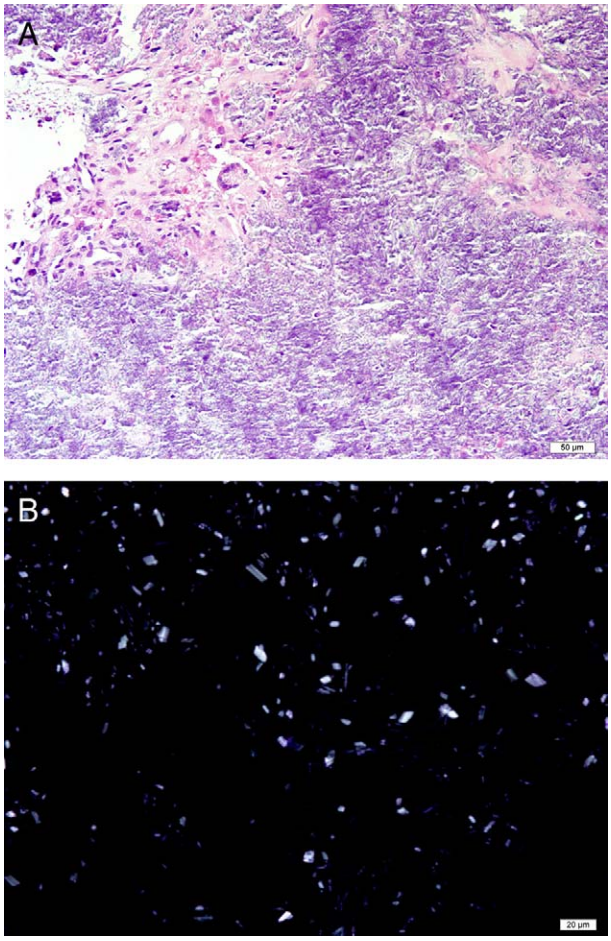

Fig. 3. (A) Histological examination of the specimen shows abundant crystal depositions in fibrous tissue (haematoxylin–eosin). (B) Under polarized light these crystals demonstrated positive birefringence.

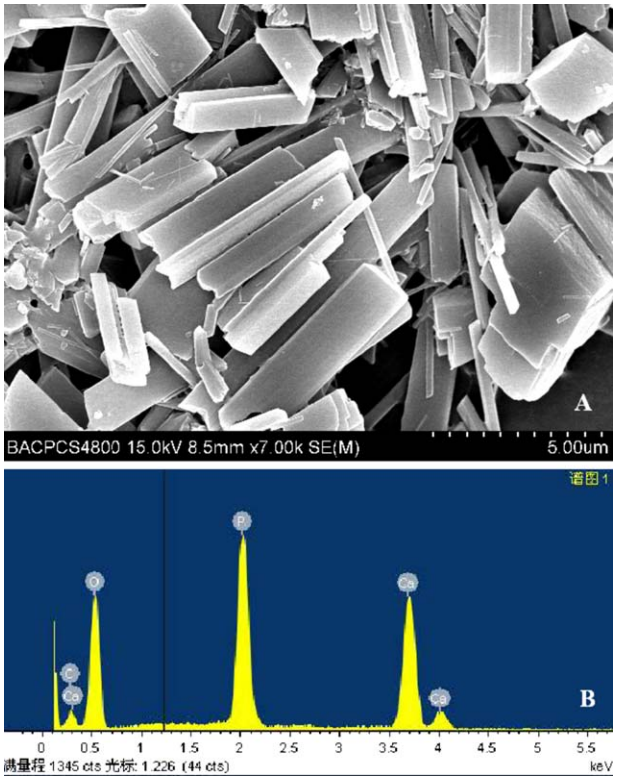

Fig. 4. (A) Scanning electron photomicrograph of CPPD crystals. (B) Energy dispersive X-ray spectrum, showing calcium and phosphorous peaks. P: phosphorus; Ca: calcium; and O: oxygen.

TMJ have been described in the literature and are summarized in Table 1. The ratio of females to males was 1.8:1 (27:15), the average age was 63 years (35–85 years, median 56 years). Most of the cases were unilateral lesions, and the ratio of left to right was close to 1.5 (22:15). Two cases were bilateral, and three cases were not stated. The common clinical manifestations include pain (30/42), swelling or mass (31/42), trismus or limitation of condylar movement (19/42), hearing loss or deafness (6/42) and malocclusion (3/42). Radiologically, calcified, radiopaque or dense masses (32/41) were most commonly found in the joint space, surrounding the condyle or extending into neighbouring tissues. Over half of the cases (27/41) showed bony changes to different extents, from sclerosis and erosion of articular cortex to severe osseous destruction. Five cases demonstrated destruction of the skull base and two extended into the middle cranial fossa.

The treatment modalities were predominantly surgical. Thirty-five cases underwent surgery, including excision (31 cases), partial excision (one case), biopsy (two cases) and irrigation (one case). Medication was prescribed in six cases, and one had no treatment. Follow-ups over 6

Table 1. Summary of the published cases of CPPD crystal deposition disease in TMJ.

Author	Side	Age	Sex	Clinical findings	Radiological findings	Treatment modalities	Recurrence (follow-up)
Pritzker et al.[36]	R	55	M	Painless mass	Radiopaque mass, articular erosion	Excision	No (2 yr)
de Vos et al.[6]	L	51	F	Chronic pain, deviation of mandible	Calcified mass on leading edge of condyle	Excision	No (1 yr)
Good & Upton[11]	L	56	M	Painful swelling	Flattened condyle, sclerosis of articular surfaces	Medication	No (2 yr)
Zemplenyi & Calcaterra[41]	L	51	F	Painful swelling, trismus	Dense mass between condyle and coronoid process, no bony destruction	Excision	Yes (2 yr)
Kamatani et al.[20]	L	57	M	Malocclusion	Dense mass between condyle and coronoid process, hypertrophic condyle	Excision	NA
Gross et al.[15]	L	59	F	Painful joint, trismus	Destructive changes in condylar head	Excision	No (10 mo)
Mogi et al.[28]	R	54	F	Pain, swelling and trismus	Irregular changes to fossa, calcified mass	Excision	No (20 mo)
Hutton et al.[16]	R	78	F	Painful joint, trismus	No abnormality	None	NA
Hutton et al.[16]	R	76	F	Acute ear pain, trismus	Calcified mass in joint space, loss of normal condylar shape	Medication	NA
Hutton et al.[16]	R	68	F	Acute pain, trismus	Nonspecific changes	Medication	NA
Lambert et al.[23]	R	41	M	Chronic painless mass, restriction of movement and deafness	Calcified mass around condyle, erosion and sclerosis of zygomatic process, temporal bones, destruction of skull base	Excision	NA
magno et al.[24]	L	53	F	Ear pain with deafness	Calcareous masses in TMJ, erosion into temporal bone, irregular condyle	Excision	No (2 yr)
dijkgraaf et al.[7]	L	53	F	Acute painful swelling, trismus, malocclusion	Calcified material in joint space, lysis of the condyle and articular eminence	Excision	1st – yes (11 mo); 2nd – no (22 mo)

Table 1 (*Continued*)

Author	Side	Age	Sex	Clinical findings	Radiological findings	Treatment modalities	Recurrence (follow-up)
CHUONG & PIPER[4]	B	65	F	Painful swelling, trismus	Abnormal signal intensity within joint space, severe osteoarthrosis	Excision	No (2 yr)
PYNN et al.[37]	L	58	M	Painless swelling and malocclusion	A cloudy and diffuse radiopacity, flattened and sclerotic articular surfaces	Excision	No (3 yr)
ISHIDA et al.[17]	NA	47	F	Painless mass	Calcified lesion, erosion of condyle	Excision	NA
ISHIDA et al.[17]	NA	50	F	Painless swelling	NA	Excision	Yes (2 yr)
ISHIDA et al.[17]	NA	55	F	No symptoms	Tumourous mass in ITF and TMJ	Excision	NA
ONODERA et al.[34]	L	48	F	Painful swelling, trismus	Radiopacity around the area of TMJ	Excision	No (18 mo)
KURIHARA et al.[22]	R	85	M	Painful swelling	A calcified mass protruding from the joint space	Excision	No (6 mo)
JORDAN et al.[18]	R	80	M	Hearing loss, middle ear effusion	Mottled mass involving temporal bone, skull base and middle cranial fossa, indenting temporal lobe	Excision	NA
STROBL et al.[40]	L	51	F	Pain and trismus, deviation of mandible	Irregular radiopaque mass around condyle, erosion of condyle	Excision	NA
GOUDOT et al.[12]	L	63	F	Painful swelling	A calcified mass filling joint space and destroying the roof of joint	Excision	No (1 yr)
NAKAGAWA et al.[29]	R	60	F	Painful swelling, restricted mouth opening	A large calcified mass around condyle, extending into ITF, erosion and sclerosis of condyle	Excision	No (3 yr)
NAKAGAWA et al.[29]	L	45	F	Pain	Faint calcification in joint space	Medication	NA
NAKAGAWA et al.[30]	R	76	M	Painful swelling, limitation of mouth opening	Joint effusion in joint space on MRI, no bony abnormality	Irrigation	No (18 mo)
AOYAMA et al.[1]	L	45	F	Painful swelling	Radiopaque images around TMJ, no destructive bony changes	Excision	No (7 mo)
ERIKSSON et al.[9]	R	72	M	Painful swelling	A well-defined mass with heterogenous signal intensity, sclerosis of condyle	Excision	No (5 yr)
OLIN et al.[32]	L	51	F	Painless swelling	Radiodense in joint, osseous destruction of sphenoid	Partial excision	No (18 mo)
GREAVES & FORDYCE[14]	B	56	M	Bilateral painful swelling, trismus	Calcification within both TMJs, sclerosis and flattening of articular surfaces	Medication	NA
COTTRELL et al.[5]	R	68	F	Swelling and nontender mass	Large gritty and lobular radiopaque mass in joint space, around the condyle	Excision	No (1 yr)
OSANO et al.[35]	L	40	M	Painful swelling	Severe destruction of the condyle	Excision	No (2 yr)
MARSOT-DUPUCH et al.[25]	R	70	M	Painful swelling, hearing loss	A calcified soft tissue with osseous remodelling, joint space widening	Biopsy	NA
MARSOT-DUPUCH et al.[25]	L	53	F	Acute aural fullness and conductive hearing loss	A large mass in glenoid fossa, eroding into middle cranial fossa	Biopsy	NA
GOLDBLATT et al.[10]	R	57	M	Severe pain and swelling	Enlarged and irregular condyle, calcified mass, articular sclerosis and erosions	Medication	NA
DIMITROULIS[8]	L	44	F	Persistent pain, trismus, and intermittent swelling	Perforated glenoid fossa, calcified specks within disc, condylar sclerosis	Excision	No (6 mo)
SMOLKA et al.[39]	L	74	F	Painful swelling	Calcified mass in joint space, no destructive bony changes	Excision	No (1 yr)
CASCONE et al.[3]	L	64	M	Preauricular swelling, trismus	An amount of calcified material around the condyle	Excision	No (5 yr)
NAQVI et al.[31]	L	35	M	A painful mass in TMJ area, tinnitus, along with hearing loss	A calcified mass around condyle, extending into ITF, eroding anterior wall of epitympanum and the skull base;	Excision	NA
REYNOLDS et al.[38]	L	52	F	Painful swelling, limited mouth opening	Distending of joint space and soft tissue material fillings, erosion of joint cortex	Excision	No (2 yr)
ASCANI et al.[2]	R	72	F	Painful mass of TMJ with progressive trismus	An calcified mass; destruction of condyle and the skull base; disc atrophy	Excision	No (7 mo)
KALISH et al.[19]	L	71	F	Facial pain, trismus and a large mass	Mass in ITF, sclerosis of articular eminence and condyle	Excision	No (18 mo)

Abbreviations: R, right; L, left; B, bilateral; M, male; F, female; NA, not available; ITF, infratemporal fossa; yr, year; and mo, month.

months were described in 27 cases (from 6 months to 5 years). Amongst them, recurrence was clear in three cases (from 11 months to 2 years)[7,17,41] and the others had no evidence of recurrence.

The pathogenesis of crystal formation in CPPD crystal deposition disease and its precipitation remain unclear. It is thought to be a metabolic disease associated with periarticular and intra-articular chondrocalcinosis. Prevalence increases with advancing age and the presence of metabolic/endocrine abnormalities, such as hyperparathyroidism, hypothyroidism, and hypomagnesaemia, and familial hyperphosphataemia[27].

Since it rarely involves the TMJ, CPPD deposition disease is not easily considered in the differential diagnosis of temporomandibular disorders. CPPD could mimic a symptomatic temporomandibular disease with preauricular pain and limitation of mouth opening because of the nonspecific symptoms, but swelling in the TMJ region, especially in elderly women, should be investigated further. CPPD manifested as a preauricular swelling could also mimic a parotid tumour[5,32,41] or a secondary infection of the TMJ. CT and MRI help to reveal the origin.

It is difficult to differentiate the lesion from a benign or malignant tumour of the TMJ on the clinical and radiographic findings, like chondroma or chondrosarcoma, particularly when it has extensive destruction of the glenoid fossa and the condyle, extends into the infratemporal fossa and intracranial fossa[18,19,25,29]. For many cases in the literature, including the present case, possible malignancy was the preoperative diagnosis. Bone scanning excludes metastasis. Preoperative fine-needle aspiration using CT guidance of the mass or frozen section specimens help to diagnose CPPD and avoid unnecessary radical excisions. The histological findings discriminate chondrosarcoma from CPPD, the former showing a tumour exhibiting cartilaginous tissue proliferation with cellular pleomorphism, nuclear hyperchromasia and myxoid changes in the matrix[33].

The differential diagnosis of CPPD in the TMJ should also include pigmented villonodular synovitis (PVNS) and synovial chondromatosis, two benign lesions with aggressive clinical features that rarely occur in the TMJ. PVNS can be diagnosed by characteristic MRI findings, which have very low signal intensity on both T1W and T2W sequences due to the paramagnetic effect attributed to haemosiderin pigmentation[21]. The finding of multiple radiopacities in the TMJ region may raise suspicion of a synovial chondromatosis, but in the latter multiple small ring-like or tubular signals can be seen on PD and T2-weighted images with large amounts of fluids[26]. The presence of crystal deposits that are birefringent under polarized light support the diagnosis of CPPD.

Deposits of calcium hydroxyapatite can also cause a destructive and invasive mass containing weakly birefringent crystals[13]. Some other crystals, such as calcium oxalate and synthetic steroids are also birefringent. The differential diagnosis should be based on a quantitative analysis of crystals or observation of the crystal structure[1]. The scanning electron microscopy with energy dispersive x-ray spectroscopy is a rapid method to differentiate these different crystals[31].

As its pathogenesis is unclear, there is no definitive treatment for CPPD. The most common modality in the TMJ in the literature is arthrotomy. Most patients required surgery because of extensive crystal deposits, and a few were performed because of an open exploration and biopsy.

Since crystal deposits may amplify the degenerative process and stimulate secretion of cellular proteases to clear the joint, it is proposed that treatment of CPDD should be based on prevention of crystal formation, dissolution of crystals and decreasing the biological consequences of crystal–cell interactions. Some authors suggest lavage of the joints or repeated aspiration with injection of intra-articular hyaluronan for these patients[25]. For patients with extensive crystal deposits in the joint and adjacent structures, surgical excision of the calcified mass should be performed to improve joint functions. Treatment using non-steroidal anti-inflammatory medications has been reported, and aspirin, steroids and colchicine appear to be helpful in alleviating acute arthritic attacks[37].

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Ethical approval

Not required.

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Address:
Xuchen Ma
Center for TMD & Orofacial Pain
Peking University School and Hospital of Stomatology
22 ZhongGuanCun South St.
HaiDian District
Beijing 100081
China
Tel: +86 10 62179977x5345;
Fax: +86 10 62136628
E–mail: kqxcma@bjmu.edu.cn

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